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NARRATIVE REVIEW

Echocardiography guided management of atrial fibrillation

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Abstract

Background: The growing burden of atrial fibrillation (AF) experienced by the general population translates into an increased incidence in the intensive care setting, further aggravated by illness severity. New onset AF has been established as an independent mortality predictor. Cardiology management guidelines are based on major trials that included ambulatory patients with varying degrees of ventricular systolic and diastolic dysfunction, and with variable dependences of left ventricular filling on atrial systole. Emphasis is placed on rate control combined with anticoagulation therapy, along with careful consideration of limiting any myocardial depression by antiarrhythmic medication in patients who often already have some form of structural heart disease.

Design: Narrative review Objectives: Critical care echocardiography (CCE) is well established as a widely available diagnostic and monitoring tool in haemodynamically unstable patients. It assists in identifying risk factors associated with arrhythmias, reveals parameters associated with arrhythmia chronicity, and guides therapy to facilitate a return to sinus rhythm. CCE helps guide the crucial management decision to seek either rhythm or rate control and, with rhythm control, monitors return of mechanical sinus rhythm with left atrial recovery post cardioversion. Echocardiography can also help when conflicting management goals are present, such as guideline-driven therapeutic anticoagulation in the intensive care patient that is at significant risk of bleeding.

Results: This review seeks to assist intensive care practitioners managing patients with AF, with a focus on the many benefits CCE offers, blending specific intensive care medicine data to current cardiology guidelines on arrhythmia management in these severely ill patients.

Keywords: Atrial fibrillation, Supraventricular arrhythmia, Echocardiography, Cardioversion, Rhythm control, Rate control

Introduction

The rising prevalence of arrhythmias places an increasing burden on critical care systems worldwide. Supraventricular arrhythmias, particularly atrial fibrillation (AF), are the most commonly encountered arrhythmias, affecting up to 25% of individuals over 40 years of age [1]. The incidence increases with age, illness severity, and surgical invasiveness, with perioperative AF rates reaching 15–40% after cardiac surgery and up to 46% in intensive care unit (ICU) patients with septic shock [2–6].

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AF is an independent risk factor for mortality in the critically ill [4]. New-onset atrial fibrillation (NOAF) raises a logical question as to whether its deleterious effect could be reversed by cardioversion and return to sinus rhythm (SR). There is limited data on the acute benefits of a rhythm control vs. rate control strategy [7–10], although similar outcomes have been documented over long-term follow-up [11]. Non-sustained arrhythmias may spontaneously terminate and require observation only whilst the decision for electrical (DC) or pharmacological cardioversion of NOAF remains debatable [12].

Major trials from the cardiology setting have failed to demonstrate a benefit of a rhythm control over a rate control strategy [13–15]. However, most cardiology randomised controlled trials fail to consider the degree of pre-existing left ventricular (LV) systolic and diastolic dysfunction, which can be of paramount importance in the acutely decompensated patient [13, 16–21]. Indeed, there may be relevant differences in responses to antiarrhythmic therapy in subgroups stratified by pre-existing cardiac function. In the presence of clinical equipoise regarding the best strategy, there is a physiological rationale for the use of echocardiography to help determine whether rhythm control is more favourable in certain populations [22].

Critical care echocardiography (CCE) has become the imaging modality of choice for investigating haemodynamically unstable patients in the ICU. In addition, CCE has a unique and important role in the assessment of cardiac structure, function, risk stratification, and in guiding the management of an arrhythmia [1, 8, 15, 23–25].

In this review, we illustrate the role of CCE in guiding the therapeutic management of the ICU patient presenting with NOAF or an AF of unknown duration in determining whether there is a preference for rhythm versus rate control, antiarrhythmic drug selection, guidance of DC cardioversion, anticoagulation, and the role in immediate follow-up. We also address the limitations of CCE and its parameters in the presence of AF. For literature search details, see Table 1 ESM.

Associated heart disease

Left ventricular systolic function

A reduction in LV systolic function increases the incidences of both atrial and ventricular arrhythmias. While the prevalence of AF ranges between 10 and 15% in patients with moderately reduced LV ejection fraction (LVEF), it reaches 49% in those with severe LV dysfunction [14, 19]. In a cohort of non-cardiac critically ill patients, NOAF was associated with a LVEF < 35% [26].

Take-home message

Critical care echocardiography is an invaluable tool for management of atrial fibrillation and other supraventricular arrhythmias in intensive care. Left atrial two-dimensional static and functional parameters together with mitral pulsed wave Doppler can help decide between a rhythm versus rate control strategy for arrhythmia management.

Right ventricular function, pulmonary hypertension and cor pulmonale

Right ventricular (RV) dilatation is associated with new-onset arrhythmias [27]. Across all forms of pulmonary hypertension (PH), the prevalence of AF ranges from 26 to 31% [28, 29]. Arrhythmias are often a harbinger of clinical deterioration with a worsening of RV failure symptoms in the majority of PH patients [30]. Right atrial (RA) dilatation with spherical remodelling and elevated RA pressure (RAP) are associated with a high prevalence of AF. In mechanically ventilated patients with septic shock, the presence of PH following cardioversion, defined as systolic pulmonary artery pressure (SPAP) ≥ 51 mmHg obtained by echocardiography, was associated with multiple recurrences of AF [25, 30]. Cardiology guidelines suggest the echocardiographic assessment of PH by the peak tricuspid pressure gradient without considering the RAP estimated from the lower vena cava diameter and its respiratory variation [31, 32]. However, most ICU patients have a central venous catheter allowing for direct measurement of the RAP and more precise SPAP estimates.

Acute cor pulmonale (RV dilatation associated with paradoxical septal motion) may trigger AF contributing to haemodynamic instability. In chronic PH, the RV typically develops adaptive wall hypertrophy, allowing it to generate increased systolic pressure to counteract an elevated afterload. In the setting of acutely reduced RV contractility, NOAF or high-rate tachycardia can rapidly result in RV failure and cardiogenic shock [33].

Diastolic function and left atrium

LV diastolic dysfunction is common in ICU patients. It is associated with a median age of 55–65 years with inherent physiological alteration of LV relaxation, prevalence of hypertension, various forms of heart failure, ischaemic heart disease, and sepsis [34]. In patients with AF, the mitral A wave is missing; however, with intermittent cardioversion, the spectral mitral Doppler profile may provide valuable insights on prognosis [35] and on the dependence of LV filling on LA contraction [25, 36, 37]. For example, patients with abnormal (prolonged) relaxation and "normalised" (type II) mitral Doppler profile [22] experience greater benefit from maintenance of SR when

exposed to critical illness than patients with advanced LV diastolic dysfunction reflected by a restrictive (type III) mitral Doppler pattern [22], where rate control may be more appropriate. Interestingly, no cardiology trial has yet attempted to stratify patients according to the severity of LV diastolic dysfunction (hence to the degree of contribution of atrial contraction to LV filling) when evaluating the management strategies (rhythm versus rate control) in the non-critically ill. Surprisingly, there is a noticeable knowledge gap on predictors of successful cardioversion in critically ill patients with AF, despite its high incidence and significant relationship with survival [25, 36–38]. An understanding of the underlying pathophysiology may be a guide to more effective treatment.

The LA dilates progressively in patients with LV diastolic dysfunction, reflecting the increased contribution of LA contraction to filling of the poorly relaxing (stage I) and progressively less compliant (higher stages) LV. Restrictive LV filling with associated elevated atrial pressure promotes AF; conversely, the LA dilatation may be a consequence of prolonged AF. Enlarged LA size (area or volume) and LV diastolic dysfunction were repeatedly identified as independent predictors of NOAF in ICU patients [38–40].

AF is typically associated with an increased LA size, as reflected by the elevated end-systolic LA diameter (LA_ESD) and biplanar end-systolic LA volume indexed to body surface area (LAVI, Fig. 1 ESM; Fig. 1). The LA_ESD should not be larger than 45 mm [41, 42]. However, a value ≥ 46 mm measured with TTE poorly predicts recurrent AF in patients with septic shock, and the LAVI should be preferred (Fig. 1) [25]. Irrespective of the parameter used to assess LA, TOE has not been validated for this measurement since the size of the anatomical structure exceeds the width of the two-dimensional field [43, 44].

Normal LAVI should be less than 35 ml/m^2 in SR and less than 40 ml/m^2 in patients with AF. The 35 ml/m^2 cut-off reflects elevated LV end-diastolic pressure and corresponds to a diagnostic criterion of LV diastolic

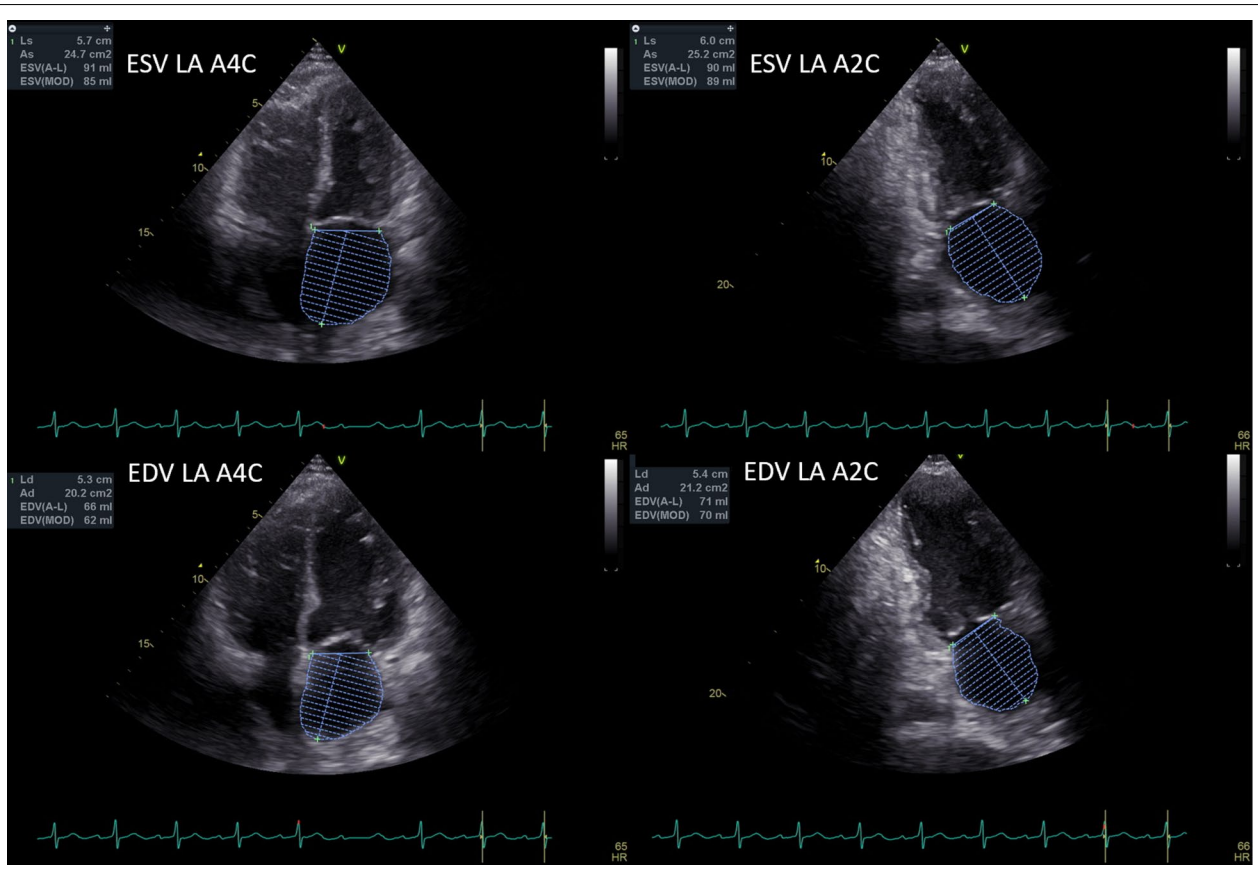

Fig. 1 Measurement of both the left atrial volume indexed to body surface area and left atrial ejection fraction 4 h after a successful cardioversion. The end-systolic volume in the apical four-chamber view (91 mL, left upper panel) is averaged with the end-systolic volume in the apical two-chamber view (90 mL, right upper panel) and divided by the body surface area of 2 m^2 to produce an indexed left atrial volume of 45 mL/m^2. Left atrial volumes at end-diastole (66 mL in the apical four chamber view, left lower panel; 71 mL in the apical two chamber view, right lower panel) allow computing a left atrial emptying fraction of 24%

dysfunction [22, 31]. A large LAVI may predict a long-standing or recurrent AF, and potentially guide clinicians towards rate rather than rhythm control [39, 45–49].

Valve disorders

Valvulopathies are independently associated with arrhythmias [1, 50]. About one-third of patients with AF have associated valvular disease, irrespective of its severity as assessed by echocardiography [1, 51]. Arrhythmia is a mortality predictor in valvular disease signalling cardiac decompensation due to pressure and/or volume overload. Accordingly, the development of arrhythmias in a patient with severe valvular disease may trigger valve repair or replacement. Alternatively, the presence of valvular disease may indicate the chronicity of a recently identified arrhythmia [14]. Current terminology dedicates the term "valvular aetiology" of arrhythmia to either rheumatic heart disease, typically mitral stenosis, or to an arrhythmia occurring in patients with mechanical prosthetic valves. A diagnosis and grading of valvular disease may help estimate the risk of intracardiac thrombus formation. An arrhythmia with a stenotic valve, especially mitral stenosis, increases such risk. In contrast, the presence of a moderate-to-severe MR reduces the risk of LA clotting due to the washout effect associated with volume recirculation between atrium and ventricle [50, 52].

Echocardiographic assessment during AF

The ventricular systolic function parameters show an acceptable intra-observer and inter-observer variability if 3–5 cardiac cycles are averaged in the presence of AF, or when measuring an index beat where the two preceding RR intervals are equal [23]. The validity and reproducibility of measurements depend on the HR and regularity of the ventricular response. Unfortunately, in addition to excluding patients with significant valvular disease, most of the published comparisons between echocardiographic and invasive measurements have been based on patients with a HR below 100/min [23]. The LVEF (e.g., biplanar Simpson´s method), eyeballing contractility estimate, mitral annular plane systolic excursion (MAPSE), or mitral tissue velocity S´ may help estimate the probability of a recurrent arrhythmia and decide on the use of an antiarrhythmic agent (Fig. 2).

Loss of the atrial kick and irregular ventricular contraction can dramatically reduce RV stroke volume, decrease SPAP, impair LV filling already compromised by the paradoxical septal motion, and decrease cardiac index [28, 30]. PH may not be detected. Altered coupling between the RV and pulmonary artery is reflected by a ratio of TAPSE to SPAP lower than 0.3–0.4, which is usually accompanied by an elevation of RAP and associated systemic venous congestion [33, 53, 54].

LAVI is one of the diagnostic criteria for LV diastolic dysfunction and is integrated in the assessment of LV preload. In addition, recent guidelines recommend the use of multiple Doppler parameters for diagnosis of altered diastolic function under AF [31, 55]. An elevated ratio (E/e´ > 11) of mitral inflow and annulus displacement at the septal aspect associated with a LAVI > 34 ml/m^2 predicts the occurrence of AF [39, 46]. Every 1 ml/m^2 increment in LAVI increases the risk of recurrent AF by 21% [46].

Both congestion and hypovolemia of the left heart chambers result in arrhythmias in ICU patients. This arrhythmogenic tendency may be further exacerbated by catecholamine infusion, which may precipitate NOAF [26, 56]. The irregular LV stroke volume observed during AF results from the variable diastolic filling time and thus constitutes a limitation in the use of Doppler analysis, including following a fluid challenge or a passive leg raising test to evaluate fluid responsiveness (Table 1 ESM) [57]. Stroke volume or its surrogate, the left ventricular outflow tract velocity–time-integral, remains meaningful endpoints to assess the response to volume expansion, but averaging several consecutive cardiac cycles is mandatory to minimise beat-by-beat variability. For the same reasons, respiratory variations of maximal aortic Doppler velocity in ventilated patients cannot be used as an index of fluid responsiveness [58]. In contrast, the collapsibility index of the superior vena cava remains valid in ventilated patients with AF, since the loss of SR does not interfere with interactions between these great vessels and the lungs during the respiratory cycle. The position of the interatrial septum during the respiratory and cardiac cycles can outperform the Doppler indices of preload in an unstable patient with AF [59].

Recent cardiology guidelines suggest that the following Doppler parameters indicate elevated LAP: E > 1 m/s, E wave deceleration time (DtE) < 160 ms, septal E/e' > 11, tricuspid regurgitation velocity > 2.8 m/s. Therefore, they can be used to contraindicate fluid therapy, especially if bilateral pulmonary B-lines are simultaneously recorded by lung ultrasound [31, 55]. Unfortunately, usual Doppler indicators are influenced by tachycardia and rhythm irregularity during AF, afterload, and mechanical ventilation, all of which complicate their interpretation for preload assessment in the critically ill. When estimating the LV filling pressure, the correlation coefficients between the invasive measurements used as reference and mitral Doppler indices (E, DtE, E/e´) range between 0.4 and 0.7 across observational studies [23]. The pulmonary vein Doppler velocities (systolic wave and diastolic wave with its deceleration time, Dtd) show promise with

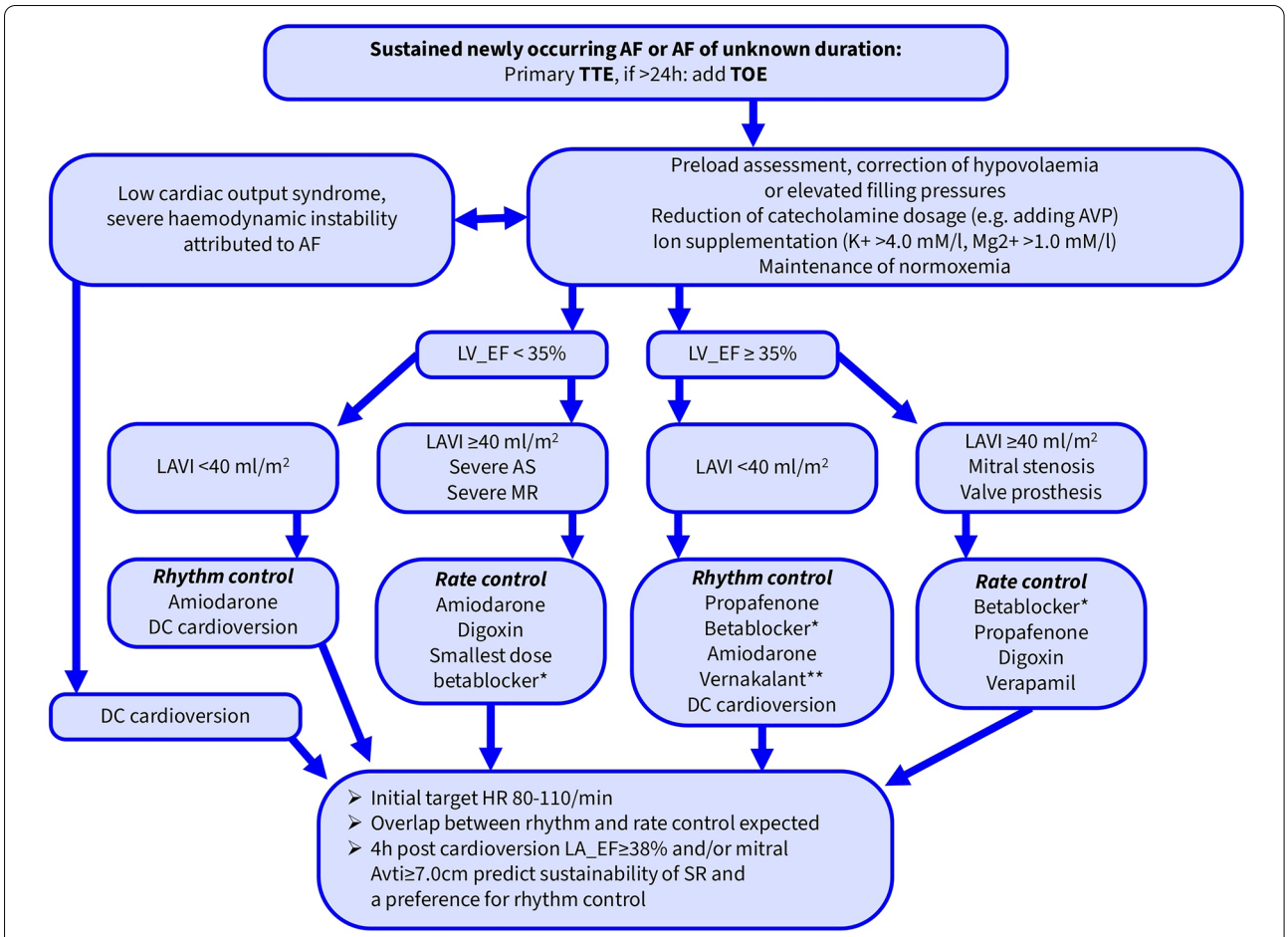
Sustained newly occurring AF or AF of unknown duration:
Primary TTE, if >24h: add TOE
Low cardiac output syndrome, severe haemodynamic instability attributed to AF
Preload assessment, correction of hypovolaemia or elevated filling pressures
Reduction of catecholamine dosage (e.g. adding AVP)
Ion supplementation (K+ >4.0 mM/l, Mg2+ >1.0 mM/l)
Maintenance of normoxemia
LV_EF < 35%
LV_EF ≥ 35%
LAVI <40 ml/m²
LAVI ≥40 ml/m² Severe AS Severe MR
LAVI <40 ml/m²
LAVI ≥40 ml/m² Mitral stenosis Valve prosthesis
Rhythm control Amiodarone DC cardioversion
Rate control Amiodarone Digoxin Smallest dose betablocker*
Rhythm control Propafenone Betablocker* Amiodarone Vernakalant** DC cardioversion
Rate control Betablocker* Propafenone Digoxin Verapamil
DC cardioversion
Initial target HR 80-110/min
Overlap between rhythm and rate control expected
4h post cardioversion LA_EF≥38% and/or mitral Avti≥7.0cm predict sustainability of SR and a preference for rhythm control

Fig. 2 Suggested algorithm for management of NOAF or AF of unknown duration, based on available intensive care medicine and cardiology data. Current evidence prefers a rhythm control for all with non-dilated LA (i.e. LAVI < 40 ml/m^2). Further steps may be facilitated according to more detailed evaluation of pre and post cardioversion parameters gathered in Table 1. Indicated lists in the boxes on pharmacotherapy start with the most preferred agents. *At the initial phase in the ICU, the short-acting beta-blockers like landiolol or esmolol are recommended. **Vernakalant may be a primary treatment if available. Abbreviations: *TTE* transthoracic echocardiography, *TOE* trans-oesophageal echocardiography, *AVP* arginine vasopressin, *LVEF* left ventricular ejection fraction, *AS* aortic stenosis, *MR* mitral regurgitation, *LAVI* indexed end-systolic left atrial volume, *HR* heart rate, *SR* sinus rhythm, *DC* direct current, *LA_EF* left atrial emptying fraction, *Avti* velocity–time integral of mitral A wave

correlation to SR reaching r = 0.80–0.91 [23, 60]. The LAP estimates obtained by deducting the peak pressure gradient of the mitral regurgitant jet from LV systolic pressure may perform well in patients with significant mitral regurgitation and AF, assuming more cardiac cycles are averaged [23].

Echocardiography guided AF treatment

Since DC cardioversion of AF is associated with a high rate of recurrence (>70%), concomitant administration of an antiarrhythmic drug is warranted in most patients [20]. However, although the success rate of the rhythm control strategy reaches 70–90%, the use of antiarrhythmic drugs is fraught with side effects, including a depressant impact on myocardial contractility [1, 20].

The cardiac depressant effect of sotalol, flecainide, and beta-blockers raises concerns when used in patients with AF [61]. Amiodarone, the least cardio-depressant antiarrhythmic agent, has been historically preferred by cardiologists treating patients with "structural heart disease" [61]. This may not be the best choice for critically ill patients with septic shock, normal-to-moderately reduced LVEF ≥ 35% and a non-dilated LA, where treatment with propafenone resulted in more frequent SR at 24 h (+16%) and less recurrence of arrhythmia (-24%) than in the amiodarone group [8, 24]. The effects of short-acting beta-blockers in AF patients with septic shock included in major trials alluded to similar benefits [62]. Current algorithms for AF management recommend against cardiac depressant medications such

as vernakalant, calcium channel blockers, propafenone, flecainide, and beta-blockers in patients with severe LV dysfunction (LVEF < 35%), for which the treatment with amiodarone and/or digoxin is reserved (Fig. 2) [1, 15].

NOAF in patients with PH is associated with a 2.1–4.7-fold increase in mortality if SR cannot be restored [63, 64]. Current guidelines advocate for a proactive rhythm-control strategy for PH patients with AF together with appropriate PH therapies [27, 32]. The priority is determining the aetiology of PH and assessment of the potential contribution of elevated LAP. The primary treatment aims at relieving RV strain by fluid unloading and adjusting mechanical ventilation to reduce RV outflow impedance. In patients with acute respiratory distress, prone positioning may also be used to unload the RV [65]. Since drugs with minimal negative inotropic activity are preferred, amiodarone is frequently used as a first-line therapy. Patients with severe RV dysfunction and preserved LV systolic function seemed to gain a larger benefit from the 1C class propafenone for rhythm control compared with amiodarone [24]. Alternative drugs which allow limiting the use of amiodarone with its potential pulmonary side effects include cautiously titrated short-acting beta-blockers (esmolol or landiolol). In case of poor tolerance due to their negative inotropic and chronotropic effects in patients with compromised RV function, infusion discontinuation will quickly restore the previous haemodynamic status. A rate control strategy (target around 100/min) is implemented if rhythm control fails. This would involve the use of amiodarone (which helps both rhythm and rate), beta-blockers (if tolerated), or digoxin.

A relationship between LA size and success of cardioversion with maintenance of SR could not be confirmed in ICU patients, unlike cardiology patients [25]. Accordingly, in haemodynamically unstable patients with low cardiac index and NOAF, a moderately dilated LA should not preclude attempts at cardioversion. Regarding specific rhythm control approaches, cardioversion with the beta-blocker derivative propafenone (class 1C antiarrhythmic) in patients with septic shock, NOAF and a non-dilated LA (LAVI < 40mls/m^2) was associated with a 12-month survival benefit compared to amiodarone (OR 0.6,p = 0.01) [8, 62]. Thus, it seems that echocardiographic assessment of LAVI may detect patients with a potential survival benefit when undergoing assessment for rhythm control and cardioversion. In AF patients, 3D TTE assessment of LA volume may also predict successful cardioversion or radiofrequency catheter ablation [66].

Cardioversion to SR may not immediately translate into measurable changes in LA function using echocardiography, especially in the presence of markedly elevated LV filling pressure and failing LA. Although LA contraction may sometimes be restored immediately after cardioversion, mechanical recovery may often be more gradual and not be seen until 4–6 h post cardioversion (Figs. 1, 3) [25, 36–38]. Patients with SR on ECG without a substantial mechanical contraction (no A wave on the mitral inflow pattern) are at high risk of arrhythmia recurrences. In such patients, a rate control strategy should be preferred for the long term rather than vain iterative attempts at restoring SR. In patients with chronic persistent arrhythmia, LA functional recovery may take up to one month or longer after cardioversion [67, 68]. The LA emptying fraction (LAEF(%): [end-systolic LAV-end-diastolic LAV] / end-systolic LAV, Fig. 1) is typically between 30–60% in a healthy person in SR. LAEF is less dependent on preload when compared to LAVI or LA diameter [41, 69]. LAEF may therefore enable better decision making in the clinical setting of abrupt changes in loading conditions seen in the critically ill. In patients with septic shock, LAEF greater than 44% was associated with a sustained SR after cardioversion, and a cut-off value of 38% at 4 h after cardioversion was predictive of SR maintenance (AUC 0.69). Lower levels (Fig. 1), however, showcased almost linear inverse relationships to the number of arrhythmia recurrences [25].

During SR, LA contraction can be semi-quantitatively assessed by the measurement of both the A wave maximal velocity and velocity–time integral (Avti, Fig. 3). In a trial focussing on rhythm control in patients with septic shock, an Avti greater than 8.6 cm at 4 h after cardioversion predicted sustainability of SR, and its value was lowered proportionally to the number of AF recurrences [25]. The main limitation of this parameter is tachycardia, which leads to the fusion of both early (E) and late (A) mitral Doppler waves. To circumvent this limitation, these measurements can be performed using the trapezoid method (Fig. 3; Fig. 2 ESM). Unfortunately, this approach is not always reliable, as demonstrated in exercising healthy volunteers when a correction for HR was suggested [70]. A HR ≥ 85/min after cardioversion could potentially increase Avti due to the fusion of the A wave with the E wave, and potentially falsely predict rhythm control. Nonetheless, this relationship of Avti to HR was not proven clinically relevant in critically ill patients with septic shock and low afterload, suggesting a possible usefulness of Avti to predict AF relapse [25].

Myocardial deformation imaging can improve the diagnostic and prognostic role of echocardiography in the management of patients with AF. LA strain determined by two-dimensional speckle tracking echocardiography is a powerful predictor of AF recurrence after catheter ablation [71]. A growing body of evidence supports the estimation of LA strain for the assessment of LA function and, potentially, also recovery after cardioversion (Fig. 3

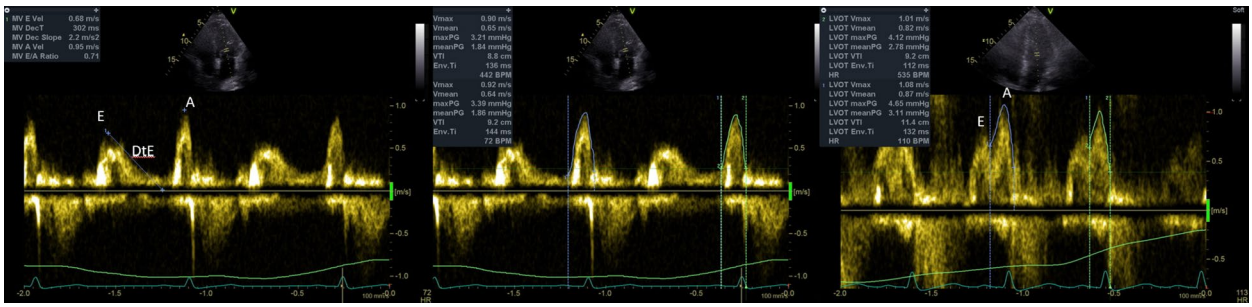

Fig. 3 Mitral Doppler pattern recorded 1 h after cardioversion (left panel) depicting a grade I left ventricular diastolic dysfunction with an E wave maximal velocity of 0.68 m/s, an E wave deceleration time of 302 ms, and an A wave maximal velocity of 0.92 m/s. Mean A wave velocity–time integral (middle panel) is 9 cm and indicates an increased contribution of LA contraction to LV filling. The right panel shows the mitral Doppler profile recorded in another patient with sinus tachycardia, resulting in partial fusion of the E and A waves. In this patient, A wave velocity–time integral reaches 10.3 cm using the trapezoid method (see also Fig. 2 ESM)

ESM) [41, 48]. The major limitations of atrial strain are the absence of ICU and pathology-specific reference values and current ICU TTE imaging quality [72].

Echocardiography-guided rhythm versus rate control approach

DC cardioversion is indicated in the management of severe haemodynamic instability and impending cardiogenic shock with low cardiac output due to arrhythmia [1, 15]. Its success, however, increases to > 80% with the addition of an antiarrhythmic agent [20].

Echocardiographic assessment (Fig. 2) starts with an estimate of LV systolic function to ensure avoidance of cardiac depressant anti-arrhythmics in patients with compromised LV systolic function. The next step is the correction of hypovolaemia in patients on vasopressors or elevated LV filling pressures that both reduce the likelihood of successful cardioversion (Table 1 ESM).

The decision regarding rhythm or rate control should follow this initial assessment (Fig. 2; Table 1). In patients with NOAF, a non-excessively dilated LA, absence of significant valvular disease, and high catecholamine requirements, the rhythm control strategy is preferred. The main reason is the immediate improvement of stroke volume and vascular resistance associated with the restoration of SR, which decreases the need for vasopressors (Fig. 4). The ensuing withdrawal of catecholamines reduces cellular stress, tachycardia, and the risk of arrhythmia recurrence [56].

Figure 4 depicts the impact of cardioversion on haemodynamic variables recorded in 187 patients with septic shock [25]. Within 4 h following the restoration of SR, both HR and cardiac output decreased despite an increase in LV stroke volume. The systemic vascular resistance rose significantly after cardioversion, restoring mean arterial pressure despite the decrease in cardiac output and allowing for a reduction in the dose of vasopressors (noradrenaline and arginine-vasopressin, Fig. 0.4). If cardiac oxygen requirements are predominantly linked to heart rate and afterload, then cardioversion is a cardiac protective step under conditions of haemodynamic stress and septic shock. The prognostic benefit is demonstrated in patients with a non-dilated LA and persistent SR after cardioversion with a mortality benefit at 1 year [8, 62]. Monitoring the effects of cardioversion is warranted especially in borderline cases, e.g. with mild-to-moderate LA dilatation (i.e. < 48 ml/m^2, Table 1) [41]. In those patients, adequate LAEF (≥ 38%) and Avti (≥ 7.0 cm) at 4 h suggest an opportunity for permanent rhythm control, whereas lower values may indicate a risk of arrhythmia recurrences [25].

In patients with significant LA dilatation, moderate-to-severe valvular disease and/or high dose of catecholamines, a rate control strategy is indicated. Due to overlap between the rhythm and rate control approaches, cardioversion may be applied in borderline cases (Table 1) [11]. The benefit of rate control lies in the extension of the diastolic filling time, allowing for improved LV filling, extended coronary artery myocardial perfusion and a reduction in myocardial oxygen consumption. To date, no major trial has demonstrated superiority of the rhythm control over the rate control strategy during the long-term observation of cardiac patients [13]. This absence of evidence cannot exclude benefits when bridging haemodynamically unstable and closely monitored patients throughout their critical illness [7, 9, 10, 62].

Echocardiography-led anticoagulation strategy

A patient with AF of unknown duration or known to exist for more than 24 h should undergo a TOE prior to DC cardioversion to rule out an underlying intracardiac thrombus and to reduce the risk of systemic

Table 1 Examples of echocardiography parameters directing intensivists to rhythm or rate control in patient with AF of unknown duration

Echocardiography parameter	Rhythm control	Uncertain – rhythm control preferred, for repeated assessment	Rate control
LAVI	<40 ml/m^2	–	>48 ml/m^2
LA_ESD	<46 mm	–	$>>46$ mm
E/e´ post CV	7–11	≥ 12 or ≤ 7	–
LAEF at 4 h post CV	$>38\%$	38–20%	$<20\%$
Avti at 4 h post CV	>7 cm	5–7 cm	<5 cm
Aortic valve	Normal-to-moderate AS, AR	–	Severe AS
Mitral valve	Normal-to-moderate MR	–	Severe MS, MR, valve prosthesis
PAPs at 4 h post CV	<51 mmHg	≥ 51 mmHg	–

LAVI left atrial end-systolic volume indexed to BSA, *LA_ESD* left atrial end-systolic diameter, *E/e´* indexed early mitral E wave to early relaxation on tissue velocity imaging of mitral annulus, *LAEF* left atrial emptying fraction, *Avti mitral* A wave velocity–time integral, *PAPs* pulmonary artery systolic pressure, *CV* cardioversion, *AS* aortic stenosis, *AR* aortic regurgitation, *MS* mitral stenosis, *MR* mitral regurgitation)

embolization during the procedure [14, 15]. This cardiology recommendation may apply to critically ill patients who often exhibit high plasma levels of multiple pro-coagulation factors but are also prone to bleeding complications. Thus, the benefit/risk balance should be carefully assessed in each individual prior to making a decision on anticoagulation. TOE is easily performed in critically ill patients who are often intubated and sedated. This exam has a sensitivity and specificity above 90% for the detection of intracardiac sludge or thrombus, especially in the LA or in the left atrial appendage (LAA) which are closest to the transducer. Spontaneous echo contrast is caused by low blood flow velocities promoting red blood cell aggregation. The swirling motion differentiates spontaneous echo contrast from an excessive gain setting [41]. Spontaneous echo contrast was observed in 20% of patients with a stroke, versus 5% in patients without stroke [73]. A thrombus may present as a mobile/sessile mass in the LAA. In addition, a LAA Doppler flow velocity <0.4 m/s indicates a risk of thrombus formation, whilst very low velocities (≤ 0.3 m/s) aggravate the clotting risk (Fig. 4 ESM) [15, 41].

In ICU patients, the risk of intracardiac thrombus formation can be stratified according to the size of the LA, the presence of valvular disease, and LV systolic function. For example, a patient in AF with normal echocardiographic findings has a 1.5% risk of intracardiac thrombus formation. Therefore, anticoagulation for an arrhythmia should be weighed against the side effects in critically ill patients in whom conditions such as recent surgery, coagulopathy, or thrombopenia are not uncommon. Moreover, the suggestion from the cardiology guidelines that the benefit of anticoagulation outweighs the risk when the estimated stroke risk is higher than 2%, calculated with an aid of available risk scores (CHA2DS2-VASc ≥ 2), can expose intensive care patients to an unacceptable risk of bleeding [14, 40, 74]. Nonetheless, echocardiography-driven risk assessment demonstrates a 20% chance of developing an intracardiac thrombus in a patient with AF, dilated LA, moderately-to-severely reduced LV systolic function, and an absence of MR [45, 52]. In addition, the risk of thrombus will further increase in the presence of valvular stenosis or a mechanical valve prosthesis. The restoration of mechanical SR should reduce requirements for anticoagulation, especially if adequate post-cardioversion LAEF and Avti are present. Thus, rhythm control may logically reduce the risk of bleeding, although it should be noted that ICU data are lacking.

Conclusion and limitations

This review adds ICU-specific data to the current cardiology guidelines on arrhythmia management to assist in the management of critically ill patients with AF. Actual major limitations include scarcity of the intensive care data, and the small sample size of the single centre studies. Echocardiography has become the modality of choice for investigating haemodynamically unstable patients in the ICU [75]. With regards to the growing burden of AF, the authors believe that measurement of the end-systolic LAVI prior to treatment and the LAEF after cardioversion, respectively predictors of the probability of success or of AF recurrence, should be implemented as part of the 2D echocardiography assessment (Fig. 1). An echocardiography-driven approach to guide AF management is outlined including considerations underpinning a rhythm or rate control strategy in the critically ill. Available data paves the way for further prospective

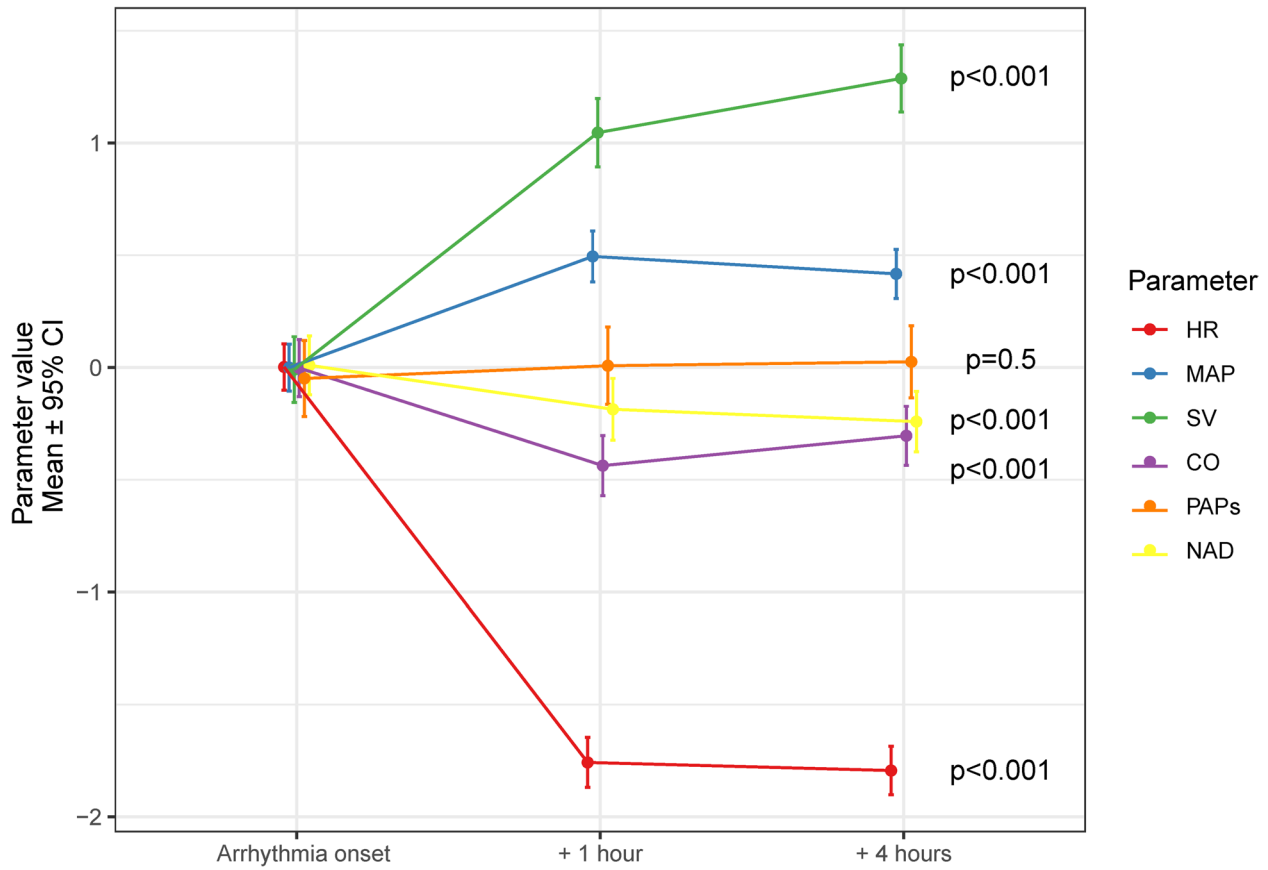

Fig. 4 Haemodynamic parameters recorded at the onset of arrhythmia, at 1 h after cardioversion, and at 4 h after cardioversion in 187 patients with septic shock and associated AF [25]. The standardised values of each parameter were obtained by subtracting the mean value at the onset from each value of that parameter (at arrhythmia onset, 1 h and 4 h after cardioversion) and dividing by the standard deviation at the onset of that parameter (median, IQR, p-values for differences at 1 h vs arrhythmia, linear mixed effect model). Abbreviations: *HR* heart rate, *MAP* mean arterial pressure, *SV* stroke volume, *CO* cardiac output, *PAPs* pulmonary artery systolic pressure, *NAD* dosage of norepinephrine

research evaluating patient-centred outcomes with echocardiography-guided arrhythmia management vs. standard of care.

Supplementary Information

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Abbreviations

AF: Atrial fibrillation; SVA: Supraventricular arrhythmia; ICU: Intensive care unit; SR: Sinus rhythm; SV: Supraventricular; AV: Atrio-ventricular; DC: Direct current; LV: Left ventricle/ left ventricular; EFLV: Ejection fraction of left ventricle; MAPSE: Mitral annular plane systolic excursion; LA: Left atrium; LAP: Left atrial pressure; LA_ESD: Left atrial end-systolic diameter; LAVI: Indexed end-systolic left atrial volume; LAEF: Left atrial emptying fraction; K^{+}: Plasmatic potassium; Mg^{2+}: Plasmatic magnesium; PAH: Pulmonary artery hypertension; PAPs: Pulmonary artery systolic pressure; RV: Right ventricle; RA: Right atrium; RAP: Right atrial pressure; TAPSE: Tricuspid annular plane systolic excursion; TTE: Transthoracic echocardiography; TOE: Transoesophageal echocardiography; E: Mitral early filling wave; DtE: Deceleration time of the E wave; A wave: Atrial contraction wave; VTI: Velocity–time integral.

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Declarations

Conflicts of interest
M.S.C. is a Section Editor for Intensive Care Medicine. She has not taken part in the review or selection process of this article. Other authors report no conflicts of interest.

Ethics approval
Not applicable.

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References

1. Hindricks G, Potpara T, Dagres N, Arbelo E, Bax JJ, Blomström-Lundqvist C, Boriani G, Castella M, Dan GA, Dilaveris PE, Fauchier L, Filippatos G, Kalman JM, La Meir M, Lane DA, Lebeau JP, Lettino M, Lip GYH, Pinto FJ, Thomas GN, Valgimigli M, Van Gelder IC, Van Putte BP, Watkins CL (2021) 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS): The Task Force for the diagnosis and management of atrial fibrillation of the European Society of Cardiology (ESC) Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC. Eur Heart J 42:373–498
2. Balik M, Matousek V, Maly M, Brozek T (2017) Management of arrhythmia in sepsis and septic shock. Anaesthesiol Intensive Therapy 49:419–429
3. Balik M, Kolnikova I, Maly M, Waldauf P, Tavazzi G, Kristof J (2017) Propafenone for supraventricular arrhythmias in septic shock-Comparison to amiodarone and metoprolol. J Crit Care 41:16–23
4. Arrigo M, Bettex D, Rudiger A (2014) Management of atrial fibrillation in critically ill patients. Crit Care Res Prac 2014:840615
5. Klein Klouwenberg PM, Frencken J, Kuipers S, Ong DS, Peelen LM, van Vught LA, Schultz MJ, van der Poll T, Bonten MJ, Cremer OL, MARS consortium (2016) Incidence, predictors and outcomes of new-onset atrial fibrillation in critically Ill patients with sepsis: a cohort study. Am J Respir Crit Care Med 195:205–211
6. Kuipers SKKP, Cremer OL (2014) Incidence, risk factors and outcomes of new-onset atrial fibrillation in patients with sepsis: a systematic review. Crit Care 18:688
7. Liu WC, Lin WY, Lin CS, Huang HB, Lin TC, Cheng SM, Yang SP, Lin JC, Lin WS (2016) Prognostic impact of restored sinus rhythm in patients with sepsis and new-onset atrial fibrillation. Crit Care 20:373
8. Waldauf P, Porizka M, Horejsek J, Otahal M, Svobodova E, Jurisinova I, Maly M, Brozek T, Rulisek J, Trachta P, Tencer T, Krajcova A, Duska F, Balik M (2024) The outcomes of patients with septic shock treated with propafenone compared to amiodarone for supraventricular arrhythmias are related to end-systolic left atrial volume. Eur Heart J Acute Cardiovasc Care 13(5):414–422
9. Si J, Ding Z, Chen X, Bai L, Sun Y, Zhang X, Zhang Y, Xia Y, Liu Y (2025) Effects of rhythm control for atrial fibrillation on cardiac remodeling and valvular regurgitation in patients with heart failure. Cardiovasc Drugs Ther 39:85–96
10. Weng C, Lin J, Liu Q, Zou CH, Zheng M, Jiang T, Jiang L, Zhuang XF, Feng H (2025) Comparison of rhythm and rate control medications for new-onset atrial fibrillation in septic patients: MIMIC-IV database analysis. J Transl Med 23:512
11. Gillinov AM, Bagiella E, Moskowitz AJ, Raiten JM, Groh MA, Bowdish ME, Ailawadi G, Kirkwood KA, Perrault LP, Parides MK, Smith RL 2nd, Kern JA, Dussault G, Hackmann AE, Jeffries NO, Miller MA, Taddei-Peters WC, Rose EA, Weisel RD, Williams DL, Mangusan RF, Argenziano M, Moquete EG, O'Sullivan KL, Pellerin M, Shah KJ, Gammie JS, Mayer ML, Voisine P, Gelijns AC, O'Gara PT, Mack MJ (2016) Rate control versus rhythm control for atrial fibrillation after cardiac surgery. N Engl J Med 374:1911–1921
12. Wetterslev M, Møller MH, Granholm A, Hassager C, Haase N, Aslam TN, Shen J, Young PJ, Aneman A, Hästbacka J, Siegemund M, Cronhjort M, Lindqvist E, Myatra SN, Kalvit K, Arabi YM, Szczeklik W, Sigurdsson MI, Balik M, Keus F, Perner A (2022) Management of acute atrial fibrillation in the intensive care unit: an international survey. Acta Anaesthesiol Scand 66:375–385
13. Al-Khatib SM, Allen LaPointe NM, Chatterjee R, Crowley MJ, Dupre ME, Kong DF, Lopes RD, Povsic TJ, Raju SS, Shah B, Kosinski AS, McBroom AJ, Sanders GD (2014) Rate- and rhythm-control therapies in patients with atrial fibrillation: a systematic review. Ann Intern Med 160:760–773
14. Ko D, Chung MK, Evans PT, Benjamin EJ, Helm RH (2025) Atrial fibrillation: a review. JAMA 333:329–342
15. Van Gelder IC, Rienstra M, Bunting KV, Casado-Arroyo R, Caso V, Crijns H, De Potter TJR, Dwight J, Guasti L, Hanke T, Jaarsma T, Lettino M, Løchen ML, Lumbers RT, Maesen B, Mølgaard I, Rosano GMC, Sanders P, Schnabel RB, Suwalski P, Svennberg E, Tamargo J, Tica O, Traykov V, Tzeis S, Kotecha D (2024) 2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J 45:3314–3414
16. Wyse DG, Waldo AL, DiMarco JP, Domanski MJ, Rosenberg Y, Schron EB, Kellen JC, Greene HL, Mickel MC, Dalquist JE, Corley SD (2002) A comparison of rate control and rhythm control in patients with atrial fibrillation. N Engl J Med 347:1825–1833
17. Van Gelder IC, Hagens VE, Bosker HA, Kingma JH, Kamp O, Kingma T, Said SA, Darmanata JI, Timmermans AJ, Tijssen JG, Crijns HJ (2002) A comparison of rate control and rhythm control in patients with recurrent persistent atrial fibrillation. N Engl J Med 347:1834–1840
18. Roy D, Talajic M, Nattel S, Wyse DG, Dorian P, Lee KL, Bourassa MG, Arnold JM, Buxton AE, Camm AJ, Connolly SJ, Dubuc M, Ducharme A, Guerra PG, Hohnloser SH, Lambert J, Le Heuzey JY, O'Hara G, Pedersen OD, Rouleau JL, Singh BN, Stevenson LW, Stevenson WG, Thibault B, Waldo AL (2008) Rhythm control versus rate control for atrial fibrillation and heart failure. N Engl J Med 358:2667–2677
19. Maisel WH, Stevenson LW (2003) Atrial fibrillation in heart failure: epidemiology, pathophysiology, and rationale for therapy. Am J Cardiol 91:2d–8d
20. Arrigo M, Jaeger N, Seifert B, Spahn DR, Bettex D, Rudiger A (2015) Disappointing success of electrical cardioversion for new-onset atrial fibrillation in cardiosurgical ICU patients. Crit Care Med 43:2354–2359
21. Echt DSLP, Mitchell LB, Peters RW, Obias-Manno D, Barker AH, Arensberg D, Baker A, Friedman L, Greene HL et al (1991) Mortality and morbidity in patients receiving encainide, flecainide, or placebo. Cardiac Arrhythm Suppress Trial NEJM 324:781–788
22. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF 3rd, Dokainish H, Edvardsen T, Flachskampf FA, Gillebert TC, Klein AL, Lancellotti P, Marino P, Oh JK, Popescu BA, Waggoner AD (2016) Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from

the american society of echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 29:277–314
23. Kotecha D, Mohamed M, Shantsila E, Popescu BA, Steeds RP (2017) Is echocardiography valid and reproducible in patients with atrial fibrillation? A systematic review. Europace 19:1427–1438
24. Balik M, Maly M, Brozek T, Rulisek J, Porizka M, Sachl R, Otahal M, Brestovansky P, Svobodova E, Flaksa M, Stach Z, Horejsek J, Volny L, Jurisinova I, Novotny A, Trachta P, Kunstyr J, Kopecky P, Tencer T, Pazout J, Belohlavek J, Duska F, Krajcova A, Waldauf P (2023) Propafenone versus amiodarone for supraventricular arrhythmias in septic shock: a randomised controlled trial. Intensive Care Med 49:1283–1292
25. Balik M, Waldauf P, Maly M, Brozek T, Rulisek J, Porizka M, Sachl R, Otahal M, Brestovansky P, Svobodova E, Flaksa M, Stach Z, Horejsek J, Volny L, Jurisinova I, Novotny A, Trachta P, Kunstyr J, Kopecky P, Tencer T, Pazout J, Krajcova A, Duska F (2024) Echocardiography predictors of sustained sinus rhythm after cardioversion of supraventricular arrhythmia in patients with septic shock. J Crit Care 83:154832
26. Makrygiannis SS, Margariti A, Rizikou D, Lampakis M, Vangelis S, Ampartzidou OS, Katsifa K, Tselioti P, Foussas SG, Prekates AA (2014) Incidence and predictors of new-onset atrial fibrillation in noncardiac intensive care unit patients. J Crit Care 29:697.e691–695
27. Tongers J, Schwerdtfeger B, Klein G, Kempf T, Schaefer A, Knapp JM, Niehaus M, Korte T, Hoeper MM (2007) Incidence and clinical relevance of supraventricular tachyarrhythmias in pulmonary hypertension. Am Heart J 153:127–132
28. Middleton JT, Maulik A, Lewis R, Kiely DG, Toshner M, Charalampopoulos A, Kyriacou A, Rothman A (2019) Arrhythmic burden and outcomes in pulmonary arterial hypertension. Front Med (Lausanne) 6:169
29. Olsson KM, Nickel NP, Tongers J, Hoeper MM (2013) Atrial flutter and fibrillation in patients with pulmonary hypertension. Int J Cardiol 167:2300–2305
30. Wanamaker B, Cascino T, McLaughlin V, Oral H, Latchamsetty R, Siontis KC (2018) Atrial arrhythmias in pulmonary hypertension: pathogenesis, prognosis and management. Arrhythm Electrophysiol Rev 7:43–48
31. Nagueh SF, Sanborn DY, Oh JK, Anderson B, Billick K, Derumeaux G, Klein A, Koulogiannis K, Mitchell C, Shah A, Sharma K, Smiseth OA, Tsang TSM (2025) Recommendations for the evaluation of left ventricular diastolic function by echocardiography and for heart failure with preserved ejection fraction diagnosis: an update from the American Society of Echocardiography. J Am Soc Echocardiogr 38:537–569
32. Humbert M, Kovacs G, Hoeper MM, Badagliacca R, Berger RMF, Brida M, Carlsen J, Coats AJS, Escribano-Subias P, Ferrari P, Ferreira DS, Ghofrani HA, Giannakoulas G, Kiely DG, Mayer E, Meszaros G, Nagavci B, Olsson KM, Pepke-Zaba J, Quint JK, Rådegran G, Simonneau G, Sitbon O, Tonia T, Toshner M, Vachiery JL, Vonk Noordegraaf A, Delcroix M, Rosenkranz S (2022) 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Heart J 43:3618–3731
33. Houston BA, Brittain EL, Tedford RJ (2023) Right ventricular failure. N Engl J Med 388:1111–1125
34. Atramont A, Lindecker-Cournil V, Rudant J, Tajahmady A, Drewniak N, Fouard A, Singer M, Leone M, Legrand M (2019) Association of age with short-term and long-term mortality among patients discharged from intensive care units in France. JAMA Netw Open 2:e193215
35. Vignon P, Charron C, Legras A, Musset F, Slama M, Prat G, Silva S, Vandroux D, Müller G, Levy B, Boissier F, Evrard B, Goudelin M, Mankikian S, Nay MA, Jabot J, Riu B, Bailly P, Maizel J, Léger J, Vieillard-Baron A (2025) Left ventricular diastolic dysfunction is prevalent but not associated with mortality in patients with septic shock. Intensive Care Med 51:94–105
36. Manning WJ, Silverman DI, Katz SE, Riley MF, Come PC, Doherty RM, Munson JT, Douglas PS (1994) Impaired left atrial mechanical function after cardioversion: relation to the duration of atrial fibrillation. J Am Coll Cardiol 23:1535–1540
37. Grundvold I, Tveit A, Smith P, Seljeflot I, Abdelnoor M, Arnesen H (2008) The predictive value of transthoracic echocardiographic variables for sinus rhythm maintenance after electrical cardioversion of atrial fibrillation. Results from the CAPRAF study, a prospective, randomized, placebo-controlled study. Cardiology 111:30–35
38. Otto C (ed) (2012) The role of echocardiography in atrial fibrillation and flutter. In: The practice of clinical echocardiography. Elsevier Health Sciences, Philadelphia, pp. 821–827
39. Fornengo C, Antolini M, Frea S, Gallo C, Grosso Marra W, Morello M, Gaita F (2015) Prediction of atrial fibrillation recurrence after cardioversion in patients with left-atrial dilation. Eur Heart J Cardiovasc Imaging 16:335–341
40. Sibley S, Bedford J, Wetterslev M, Johnston B, Garside T, Kanji S, Whitehouse T, Welters I, Ostermann M, Balik M, Lancini D, Dharmaraj B, Benjamin EJ, Walkey AJ, Cuthbertson BH, (2025) Atrial fibrillation in critical illness: state of the art. Intensive Care Med
41. Sade LE, Faletra FF, Pontone G, Gerber BLM, Muraru D, Edvardsen T, Cosyns B, Popescu BA, Klein A, Marwick TH, Cameli M, Saric M, Thomas L, Ajmone Marsan N, Fontes-Carvalho R, Podlesnikar T, Fontana M, La Gerche A, Petersen SE, Moharem-Elgamal S, Bittencourt MS, Vannan MA, Glikson M, Peichl P, Cochet H, Stankovic I, Donal E, Thomas D, Marta RS (2025) The role of multi-modality imaging for the assessment of left atrium and left atrial appendage: a clinical consensus statement of the European Association of Cardiovascular Imaging (EACVI), European Heart Rhythm Association (EHRA) of the European Society of Cardiology (ESC). Eur Heart J Cardiovasc Imaging 26:385–413
42. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, Lancellotti P, Muraru D, Picard MH, Rietzschel ER, Rudski L, Spencer KT, Tsang W, Voigt JU (2015) Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging 16:233–270
43. Block M, Hourigan L, Bellows WH, Reeves J 3rd, Romson JL, Tran M, Pastor D, Schiller NB, Leung JM (2002) Comparison of left atrial dimensions by transesophageal and transthoracic echocardiography. J Am Soc Echocardiogr 15:143–149
44. Singh H, Jain AC, Bhumbla DK, Failinger C (2005) Comparison of left atrial dimensions by transesophageal and transthoracic echocardiography. Echocardiography 22:789–796
45. Ayirala S, Kumar S, O'Sullivan DM, Silverman DI (2011) Echocardiographic predictors of left atrial appendage thrombus formation. J Am Soc Echocardiogr 24:499–505
46. Marchese P, Bursi F, Delle Donne G, Malavasi V, Casali E, Barbieri A, Melandri F, Modena MG (2011) Indexed left atrial volume predicts the recurrence of non-valvular atrial fibrillation after successful cardioversion. Eur J Echocardiogr 12:214–221
47. Abhayaratna WP, Seward JB, Appleton CP, Douglas PS, Oh JK, Tajik AJ, Tsang TS (2006) Left atrial size: physiologic determinants and clinical applications. J Am Coll Cardiol 47:2357–2363
48. Lim DJ, Ambale-Ventakesh B, Ostovaneh MR, Zghaib T, Ashikaga H, Wu C, Watson KE, Hughes T, Shea S, Heckbert SR, Bluemke DA, Post WS, Lima JAC (2019) Change in left atrial function predicts incident atrial fibrillation: the multi-ethnic study of atherosclerosis. Eur Heart J Cardiovasc Imaging 20:979–987
49. Osranek M, Fatema K, Qaddoura F, Al-Saileek A, Barnes ME, Bailey KR, Gersh BJ, Tsang TS, Zehr KJ, Seward JB (2006) Left atrial volume predicts the risk of atrial fibrillation after cardiac surgery: a prospective study. J Am Coll Cardiol 48:779–786
50. De Caterina R, Camm AJ (2014) What is "valvular" atrial fibrillation? A reappraisal. Eur Heart J 35:3328–3335
51. Yafasov M, Olsen FJ, Shabib A, Skaarup KG, Lassen MCH, Johansen ND, Jensen MT, Jensen GB, Schnohr P, Møgelvang R, Biering-Sørensen T (2024) Even mild mitral regurgitation is associated with incident atrial fibrillation in the general population. Eur Heart J Cardiovasc Imaging 25:579–586
52. Nakagami H, Yamamoto K, Ikeda U, Mitsuhashi T, Goto T, Shimada K (1998) Mitral regurgitation reduces the risk of stroke in patients with nonrheumatic atrial fibrillation. Am Heart J 136:528–532
53. D'Alto M, Marra AM, Severino S, Salzano A, Romeo E, De Rosa R, Stagnaro FM, Pagnano G, Verde R, Murino P, Farro A, Ciccarelli G, Vargas M, Fiorentino G, Servillo G, Gentile I, Corcione A, Cittadini A, Naeije R, Golino P (2020) Right ventricular-arterial uncoupling independently predicts survival in COVID-19 ARDS. Crit Care 24:670
54. Zhang H, Zhang D, Wang X, Liu Y, Lian H, Zhang Q, Zhao H, Chen X, Liu D (2023) Prognostic implication of a novel right ventricular injury score in septic patients. ESC Heart Fail 10:1205–1213
55. Robinson S, Ring L, Oxborough D, Harkness A, Bennett S, Rana B, Sutaria N, Lo Giudice F, Shun-Shin M, Paton M, Duncan R, Willis J, Colebourn C, Bassindale G, Gatenby K, Belham M, Cole G, Augustine D, Smiseth OA

(2024) The assessment of left ventricular diastolic function: guidance and recommendations from the British Society of Echocardiography. Echo Res Pract 11:16
56. Schmittinger CA, Torgersen C, Luckner G, Schröder DC, Lorenz I, Dünser MW (2012) Adverse cardiac events during catecholamine vasopressor therapy: a prospective observational study. Intensive Care Med 38:950–958
57. Bunting KV, Gill SK, Sitch A, Mehta S, O'Connor K, Lip GY, Kirchhof P, Strauss VY, Rahimi K, Camm AJ, Stanbury M, Griffith M, Townend JN, Gkoutos GV, Karwath A, Steeds RP, Kotecha D (2021) Improving the diagnosis of heart failure in patients with atrial fibrillation. Heart (British Cardiac Society) 107:902–908
58. Teboul JL, Monnet X, Chemla D, Michard F (2019) Arterial pulse pressure variation with mechanical ventilation. Am J Respir Crit Care Med 199:22–31
59. Haji DL, Ali MM, Royse A, Canty DJ, Clarke S, Royse CF (2014) Interatrial septum motion but not Doppler assessment predicts elevated pulmonary capillary wedge pressure in patients undergoing cardiac surgery. Anesthesiology 121:719–729
60. Kinnaird TD, Thompson CR, Munt BI (2001) The deceleration [correction of declaration] time of pulmonary venous diastolic flow is more accurate than the pulmonary artery occlusion pressure in predicting left atrial pressure. J Am Coll Cardiol 37:2025–2030
61. Kirchhof P, Ammentorp B, Darius H, De Caterina R, Le Heuzey JY, Schilling RJ, Schmitt J, Zamorano JL (2014) Management of atrial fibrillation in seven European countries after the publication of the 2010 ESC Guidelines on atrial fibrillation: primary results of the PREvention oF thromboemolic events–European Registry in Atrial Fibrillation (PREFER in AF). Europace 16:6–14
62. Balik M, Tavazzi G, Slama M (2025) Beta-blockers as antiarrhythmics in septic shock: a light at the end of the tunnel? Intensive Care Med 51:232–234
63. Wen L, Sun ML, An P, Jiang X, Sun K, Zheng L, Liu QQ, Wang L, Zhao QH, He J, Jing ZC (2014) Frequency of supraventricular arrhythmias in patients with idiopathic pulmonary arterial hypertension. Am J Cardiol 114:1420–1425
64. Cannillo M, Grosso Marra W, Gili S, D'Ascenzo F, Morello M, Mercante L, Mistretta E, Salera D, Zema D, Bissolino A, Fusaro E, Marra S, Libertucci D, Gaita F (2015) Supraventricular arrhythmias in patients with pulmonary arterial hypertension. Am J Cardiol 116:1883–1889
65. Zochios V, Yusuff H, Antonini MV (2024) Prone positioning and right ventricular protection during extracorporeal membrane oxygenation for severe acute respiratory distress syndrome. Asaio j 70:e119–e122
66. Montserrat S, Gabrielli L, Borras R, Poyatos S, Berruezo A, Bijnens B, Brugada J, Mont L, Sitges M (2014) Left atrial size and function by three-dimensional echocardiography to predict arrhythmia recurrence after first and repeated ablation of atrial fibrillation. Eur Heart J Cardiovasc Imaging 15:515–522
67. Dell'Era G, Rondano E, Franchi E, Marino PN (2010) Atrial asynchrony and function before and after electrical cardioversion for persistent atrial fibrillation. Eur J Echocardiogr 11:577–583
68. Thomas L (2007) Assessment of atrial function. Heart Lung Circ 16:234–242
69. Malik J, Lachmanova J, Kudlicka J, Rocinova K, Valerianova A, Bartkova M, Tesar V (2016) Left atrial dysfunction in end-stage renal disease patients treated by hemodialysis. Nephron 133:169–174
70. Chung CS, Kovacs SJ (2006) Consequences of increasing heart rate on deceleration time, the velocity-time integral, and E/A. Am J Cardiol 97:130–136
71. Yasuda R, Murata M, Roberts R, Tokuda H, Minakata Y, Suzuki K, Tsuruta H, Kimura T, Nishiyama N, Fukumoto K, Aizawa Y, Tanimoto K, Takatsuki S, Abe T, Fukuda K (2015) Left atrial strain is a powerful predictor of atrial fibrillation recurrence after catheter ablation: study of a heterogeneous population with sinus rhythm or atrial fibrillation. Eur Heart J Cardiovasc Imaging 16:1008–1014
72. Vignon P, Vaidie J, Goudelin M, Evrard B (2025) Left atrial reservoir strain: an essential tool for evaluating left ventricular diastolic dysfunction in 2025-response. Author's reply. Intensive Care Med 51:992–993
73. Watson T, Shantsila E, Lip GY (2009) Mechanisms of thrombogenesis in atrial fibrillation: Virchow's triad revisited. Lancet 373:155–166
74. Walkey AJ, Quinn EK, Winter MR, McManus DD, Benjamin EJ (2016) Practice patterns and outcomes associated with use of anticoagulation among patients with atrial fibrillation during sepsis. JAMA Cardiol 1:682–690
75. Robba C, Wong A, Poole D, Al Tayar A, Arntfield RT, Chew MS, Corradi F, Douflé G, Goffi A, Lamperti M, Mayo P, Messina A, Mongodi S, Narasimhan M, Puppo C, Sarwal A, Slama M, Taccone FS, Vignon P, Vieillard-Baron A (2021) Basic ultrasound head-to-toe skills for intensivists in the general and neuro intensive care unit population: consensus and expert recommendations of the European Society of Intensive Care Medicine. Intensive Care Med 47:1347–1367